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Original Article

Sleep-related eating disorder: a descriptive study in Chilean patients

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ABSTRACT

Objectives: We aimed to describe a group of adults diagnosed with sleep-related eating disorder (SRED) at the Sleep Medicine Center of the Pontificia Universidad Catolica de Chile.
Methods: We performed a descriptive study of 34 consecutive patients who met the criteria of the *International Classification of Sleep Disorders* for SRED evaluated during a 3-year period who did not have an eating disorder according to the criteria of the *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition. All patients had a structured clinical interview performed by a sleep specialist and completed the Beck Depression Inventory (BDI). Polysomnography (PSG) was performed when clinically indicated for ruling out other sleep-related disorders (18 patients; 52.9%). Patients' demographic and clinical data, comorbidities, and treatment response also were analyzed.
Results: Most patients were women ($n = 23$; 67.6%). The average age at the time of diagnosis was 39 ± 13.8 (17–67 years) and the latency since symptom onset was 8.3 ± 8.8 years. Most patients had several episodes per night (average, 2.6 ± 1.6; 1–8) and all except one patient had partial or total amnesia of these events ($n = 33$; 97%). Comorbidities were frequent and included insomnia ($n = 20$; 58.8%), restless legs syndrome (RLS) ($n = 16$; 47%), sleep-disordered breathing (SDB) ($n = 9$; 26%), psychiatric disorders ($n = 13$; 38.2%), and overweight or obesity ($n = 14$; 41.1%). Most patients were hypnotic users ($n = 21$; 61.7%) and reported weight-centered anxiety ($n = 23$; 67.6%). Twenty patients (58.8%) were treated with topiramate, 17 of whom had adequate symptomatic responses.
Conclusion: Our SRED patients showed female preponderance, amnesia during the episodes, association with other sleep disorders, use of hypnotics, weight-centered anxiety, and positive response to topiramate. The presence of anxiety focused on weight in most patients may be an important element in the emergence of this behavior during sleep.

1. Introduction

Sleep-related eating disorder (SRED) is a parasomnia which was first described in 1991 [1,2]. The prevalence of SRED in the general population is not well-established but has been estimated between 1% and 5% in adults; however, it could be as high as 16.7% in patients who have eating disorders [3]. This most likely underdiagnosed condition is characterized by recurrent episodes of compulsive and involuntary eating and drinking at night after falling asleep, which frequently occurs during the first half of the night. Usually patients eat highly caloric food, including inedible and unusual meal combinations, or even substances like cigarettes or cologne. Weight gain and gastrointestinal disturbances can be seen as consequences of this behavior, and patients also can get injured during the careless preparation and consumption of food. SRED generally starts in young adults, with a female predominance [4,5]. Symptoms take a chronic course and the diagnosis is clinical. The pathophysiology of SRED is unknown, though it has been suggested that the dopaminergic system might be involved or that a genetic predisposition could exist [3]. SRED can be induced by several drugs (e.g., hypnotics), such as zolpidem, tricyclic antidepressant agents, anticholinergic agents, lithium, olanzapine, quetiapine, and risperidone [5–12]. Most SRED patients have other concomitant sleep disorders, like restless legs syndrome (RLS), obstructive sleep apnea (OSA), and sleepwalking, among others [4,13,14]. In our study, we present the clinical findings in a prospective series of 34 consecutive patients diagnosed with SRED, evaluated at the Sleep Disorders Clinic of the Pontificia Universidad Catolica de Chile between June 2005 and May 2010.

2. Methods

We performed a prospective observational study of consecutive SRED patients treated at the Sleep Disorders Clinic of the Pontificia Universidad Catolica de Chile between 2005 and 2010. The study was reviewed and approved by the institutional ethics committee.

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All patients who met the criteria of the *International Classification of Sleep Disorders* for SRED were included. We excluded patients with a previous diagnosis or history compatible with other eating disorders included in the *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition, classifications (e.g., bulimia nervosa, anorexia nervosa). Patients who had evening hyperphagia with full awareness during their eating, complete recall, and ingestion of usual foods only before a sleep period were considered to have night eating syndrome (NES) and were not included in our study. All patients had a structured clinical interview, which included a sleep disorders evaluation and a psychiatric assessment; the Beck Depression Inventory (BDI), which was adapted into Spanish and validated by Bonilla et al. [15], was used for each patient. Patients also had a general physical and neurologic examination performed. Patients with symptoms suggestive of sleep-disordered breathing or periodic limb movements were studied with polysomnography (PSG). We also recorded demographic and clinical characteristics, comorbidities, and therapies used.

3. Results

A total of 34 consecutive patients with SRED evaluated at the Pontificia Universidad Catolica de Chile Sleep Disorders Clinic between June 2005 and May 2010 met the diagnostic criteria for SRED. Seven patients (20.6%) were interviewed with spouses (five cases) or parents (two cases).

3.1. General characteristics

Table 1 shows the main demographic and clinical characteristics of the group. There was a clear predominance of women (67.6%). Most patients (56%) started SRED episodes as adolescents or in early adulthood. The average age at the time of the diagnosis was 39.0 years (range, 17–67 years) and the delay to the diagnosis was 8.3 years (range, 0–37 years), according to the information provided by the patients. Two-thirds of the patients ($n = 23$; 67.6%) reported anxiety regarding their weight, At diagnosis, 6 patients (18.7%) were obese (body mass index $\geqslant 30\ kg/m^2$); and another 8 (23.5%) were overweight (body mass index between 25 and 30 kg/m^2). Two of the obese patients had undergone bariatric surgery before SRED diagnosis. Both of these patients had significant increase in weight after SRED onset, though both were previously overweight.

Table 1
Clinical and demographic characteristics of the patients.

Characteristics	
Sex	
Men	11 (32.3%)
Women	23 (67.6%)
Age (y)	39.0 ± 13.8 (17–67)
BMI (kg/m^2)	25.5 ± 4.3 (18–34)
Nighttime ingestion frequency	Every night*
No. of episodes per night	
1	10 (29.4%)
2–5	20 (58.8%)
>5	4 (11.7%)
Family history with SRED	2 (5.9%)
Amnesia of the episodes	
With amnesia	33 (97.1%)
Without amnesia**	1 (2.9%)
Partial in all	17 (50%)
Total in all	9 (26.4%)
Partial or total	7 (20.5%)
Weight gain	23 (67.6%)
Diagnosis latency (y)	8.3 ± 8.8 (0–37)
Medication	21
Benzodiazepines	13 (61.9%)
Other hypnotics	15 (71.4%)

Abbreviations: y, years; BMI, body mass index; No., number; SRED, sleep-related eating disorder.
Data are shown in numbers and percentages, except age and BMI, which are shown as average values, standard deviations, and ranges.
Medication use is expressed as percentage of the total number of patients on medication.
* One patient (woman) only had 3 ingestion episodes a month.
** One patient (woman) did not have amnesia of any of the ingestion episodes.

3.2. Episode characteristics

No patient had ever specifically consulted for night-eating episodes and only sought consultation after seeing reports on SRED in the media. Almost all patients ($n = 32$; 94.1%) reported more than one eating episode per night. All but one patient had amnesia of the episodes, which was either always partial ($n = 17$; 50%), always complete (9/34; 26.4%), or partial in some episodes and total in others in 20.5% ($n = 7$).

3.3. Use of hypnotics and amnesia

Twenty three patients (67.6%) used hypnotics (benzodiazepines only [$n = 5$], benzodiazepine receptor agonists only [$n = 4$], and a mix of both [$n = 14$]). In 4 of these patients there was a clear temporal relation between the SRED onset and the use of benzodiazepine receptor agonists, in two cases as the sole medication and in the other 2 after adding them to benzodiazepines. All patients under hypnotics had amnesia of the episodes, either complete ($n = 14$; 60.8%) or partial ($n = 9$; 39.2%). Patients who did not use hypnotics mainly had partial amnesia during the episodes ($n = 7$; 64%), though two patients had complete amnesia episodes ($n = 2$; 18%). One patient from this group had no amnesia.

3.4. Other medications

Several other medications were used by SRED patients, which included selective serotonin reuptake inhibitors, trazodone, melatonin, quetiapine, cyclobenzaprine, pregabalin, gabapentin, thyroid hormone replacement, and antidiabetic and antihypertensive agents.

3.5. Comorbidities

All but one patient ($n = 33$; 97%) had one or more additional sleep disorder (Table 2). Other comorbidities also are listed in Table 2. Depression also was a common comorbidity ($n = 13$; 38.2%). The BDI scale in these latter patients was 11.1 ± 7.45, with

Table 2
Comorbidities.

Associated sleep disorders	33
Insomnia	20 (58.8%)
PSG-documented OSA	9 (26%)
RLS	16 (47%)
Sleepwalking	4 (11.7%)
Night terrors	1 (2.9%)
Bruxism	6 (17.6%)
Medical comorbidities	
Type 2 diabetes mellitus	4 (11.7%)
Thyroid disease*	3 (8.8%)
Psychiatric comorbidities	
Depression	13 (38.2%)

Abbreviations: PSG, polysomnography; OSA, obstructive sleep apnea; RLS, restless legs syndrome.
Data are shown in numbers and percentages.
* Chronic autoimmune thyroiditis.

a range between 0 and 25 (mild depression in the Chilean validation of the scale); in the group of patients without depression the BDI scale was 5 ± 2.7, with a range between 0 and 8 (nondepressed range).

3.6. Video-PSG

Our study was performed in 18 patients (52.9%), during which they had food at their disposal in case an eating episode occurred. Four of the patients (22.2%) got up between 1 and 3 times to eat during the study; the episodes arose from stages 2 and 3 nonrapid eye movement (NREM) sleep, and eating lasted between 56 and 90 s. Sleep-disordered breathing (SDB) was seen in nine patients. It was severe in six patients (average respiratory disturbance index [RDI], 42.2 [reference range, 35.2–84.9]; average arousal index [AI] 48.4 [reference range, 29.6–70.6]), mild in two patients (average RDI, 8.51; average AI, 20), and mild rapid eye movement related in one patient (RDI, 9.2 in rapid eye movement sleep stage; AI, 23). Chronic snoring without respiratory events was observed in four patients and periodic limb movements in five patients.

3.7. Treatment

Table 3 shows clinical details of the SRED episodes and therapies. Medications were used in 26 patients (76.5%); the remaining patients refused to take medication due to a fear of adverse effects. Topiramate was used in 20 patients (58.8%) as bedtime doses, starting at 25 mg daily with 25 mg weekly increases when needed depending on clinical response. Total doses were between 25 and 200 mg daily, except for one patient who needed to take 450 mg daily; the mode was 50 mg daily. Response was considered favorable when patients or relatives reported cessation or clear reduction of the episodes, which was the case in 17 patients (85%), with an average follow-up of 15.8 ± 10.6 months (range, 3–39 months). There was no response in three patients. Six patients (30%) had to discontinue topiramate due to adverse effects, which included dizziness ($n = 4$), visual problems ($n = 1$), and worsening of preexisting depressive symptoms ($n = 1$); one of these patients had recurrence of SRED after stopping topiramate. Seven patients used dopaminergic agonists (pramipexole in 5 and ropinirole in 2 patients). In three patients (42.8%) the response to dopaminergic was favorable; there was no effect in SRED episodes in the other three patients, and the remaining patient had to withdraw the medication due to intolerance. Two patients who had both SRED and restless legs syndrome received pramipexole as first therapy, one with no SRED improvement the other with adequate response to both conditions. One patient used ropinirole and one used continuous positive airway pressure (CPAP) therapy plus ropinirole. CPAP was prescribed in the severe SDB patients and 4 of patients actually used it. Two CPAP users had no more SRED episodes and improved SDB, one stopped CPAP use due to intolerance and the other patient's SRED remained unaffected by CPAP use. One patient was successfully treated with psychotherapy; she had adequate response to topiramate, but it had to be withdrawn due to significant effects after which SRED episodes relapsed. She was then placed on bupropion due to a mood disorder with no SRED response until psychotherapy was added.

Table 3
Clinical details of the sleep-related eating disorder episodes and therapies.

	Preferred food type	N/night	Amnesia	Diagnosis latency	Weight anxiety	RLS	Topiramate	Improvement
1	Cookies	2	Total	10	No	Yes	50 mg	Yes
2	Cologne, light meals	1	Partial	16	No	Yes	50 mg	Yes
3	Bread, liquor, sodas	3–4	Total	2	Yes	Yes	25 mg	Yes
4	Candy, sodas, smokes cigarettes	1	Partial	1	No	No	0	–
5	Popcorn, smokes cigarettes	1	Partial	2	Yes	No	25 mg	Yes
6	Candy, light meals	1	Partial	10	Yes	No	200 mg	Yes
7	Cookies, fruitcake, ham	1–2	Partial	2	Yes	Yes	0	–
8	Candy, fruitcake, baby mussels	2	Partial	4	No	Yes	0	–
9	Cake, ham on bread, marmalade	3–4	Partial	10	Yes	No	25 mg	Yes
10	Candy	2	Total	5	No	No	0	–
11	Mayonnaise on bread	1	Partial	6	Yes	No	0	–
12	Ice-cream, French fries, chocolate	1	Total	7	Yes	Yes	0	–
13	Tea, bread	3–4	Partial	4	No	Yes	0	–
14	Cookie, French fries	1	Mixed	1	Yes	No	0	–
15	Candy	1	Total	2	No	No	25–50 mg	No
16	Candy, bread, cured meat	4	Partial	8	Yes	No	25–450 mg	Yes
17	Bread and fungus, cooked meals	2–3	Partial	2	No	No	25 mg	No
18	Candy	2–3	Total	1	Yes	No	25–50 mg	No
19	Cooked meals, tea	1	Total	1	Yes	No	0	–
20	Chocolate, fruitcake, fruit	4	Mixed	22	Yes	No	150 mg	Yes
21*	Candy, bread	5–6	Total	15	Yes	Yes	100 mg	Yes
22	Ice-cream	5–6	Mixed	30	Yes	Yes	0	–
23	Ice-cream, cookies, peanuts, ham	1–3	Partial	12	No	No	0	–
24	Cookies, chocolate, butter	6–8	Partial	0	Yes	Yes	75 mg	Yes
25	Candy, cookies	2	Partial	2	No	No	50 mg	Yes
26	Cookies, bread, light meal	2	Mixed	2	Yes	Yes	0	–
27	Bread with fungus	3	Partial	26	No	Yes	200 mg	Yes
28	Bread, cold sausages	2	Total	0	Yes	No	0	–
29	Candy	1–2	Mixed	8	Yes	Yes	0	–
30	Candy, light meal	2	Partial	10	Yes	Yes	25 mg	Yes
31	Candy	2	Partial	6	Yes	No	25 mg	Yes
32	Cookies, cakes, cereal bars	1**	No	8	Yes	No	25 mg	Recently started
33	Sister's light meal, tobacco	5–6	Mixed	11	Yes	Yes	50 mg	Yes
34*	Fat, candy, spaghetti	3	Mixed	37	Yes	Yes	100 mg	Yes

Abbreviations: N, number; RLS, restless legs syndrome.
* Two patients underwent bariatric surgery.
** One patient (woman) only gets up 3 times a month, 1 time per night.

4. Discussion

SRED is an NREM sleep parasomnia described in the early 1990s by Schenk et al. [1,2], though the first reports on night eating in obese patients date back to 1955 [16]. There are few studies that show SRED prevalence. Winkelman et al. [3] observed a 4.6% prevalence rate in an unselected series of 127 college students, a figure which is significantly higher among patients with eating disorders. It often is associated with sleep disorders, such as RLS, OSA, and sleepwalking [1,3,12,13]. It has been described in connection with and triggered by the use of hypnotic drugs, particularly zolpidem and with other psychotropic drugs [5–11].

SRED is a parasomnia that must be distinguished from other daytime eating disorders that also manifest at night, and specifically from NES, which encompasses symptoms that are commonly confused with those of SRED [12,17,18]. Bulimia nervosa, another daytime eating disorder, also might be considered in the differential diagnosis; these patients may have a shift-forward sleep, which could be related to their binge-purge patterns [19]. NES is considered an eating disorder and not a parasomnia. Proposed diagnostic criteria include significant food intake after dinner (at least 25% of daily calories) and awareness and recall of the evening and nocturnal eating episodes. In addition, these patients consume edible food [20,21]. NES is seen as a circadian delay of food intake, with normal circadian sleep onset [22]. Eating is involuntary in SRED, almost always with either complete or partial amnesia of the episodes; it also is associated with binge eating of inappropriate food and frequently is associated with other sleep disorders [20,21]. The use of hypnotic drugs may trigger the events and contribute to the amnesia [22–24]. However, Vinai et al. [19] recently reported a study which revealed that both of these conditions might overlap.

Our series of SRED patients is demographically and clinically similar to other reported in the literature [1,12]. It shows a clear predominance of women, with symptoms beginning in adult life mostly associated with sleeping disorders and frequently associated with psychiatric comorbidities (i.e., mood disorders). The episodes themselves characteristically occur every day in most patients, usually more than one time per night, with partial or complete amnesia of the events. During the episodes patients eat mainly foods rich in carbohydrates, but other episodes include the consumption of nonedible items like cologne.

A considerable number of our patients were obese or overweight. A more significant finding was that worry and anxiety focused on weight was present in two-thirds of our patients. In some of our patients weight-centered anxiety was a premorbid condition, a fact that eventually could shed light on the pathophysiologic mechanisms underlying this condition and also could be important in complementary treatment strategies.

All our patients were motivated to consult by information on the subject that appeared in the media, and therefore the diagnosis had significant delay in almost every patient. This finding suggests that this behavior was not seen as a reason for consultation or that it was not recognized as a medical entity by physicians. It is then reasonable to assume that SRED is an underdiagnosed condition most likely even within the sleep medicine community, and it should be systematically assessed in both sleep disorders and eating disorders patients. Furthermore, night eating and the eventual weight gain can aggravate certain comorbidities, such as diabetes mellitus or OSA.

In our case series examined herein, all but one patient had total or partial amnesia of the episodes. Although the presence of amnesia is considered essential for the diagnosis, it is not included in the current criteria for SRED diagnosis, as defined by the American Academy of Sleep Medicine [25]. Amnesia could be an integral part of the symptoms and could be based on pathophysiologic mechanisms that explain it, as in other parasomnias. However, it also could be a secondary effect of the use of hypnotic drugs, particularly benzodiazepine receptor agonists [6,10,22,23].

PSG studies are useful in SRED mainly to identify associated sleep disorders observed in almost all patients, as their treatment may be required to completely reduce or control SRED. Due to economic limitations, PSGs were performed in a little over 50% of the patients in our series. The PSG was focused on patients with high clinical suspicion of associated sleep disorders who needed diagnostic confirmation. Although patients had food at their disposition during the PSG and 22 patients studied through PSG had daily amnesic episodes, only 4 of them (22.2%) had nighttime ingestion events. This finding suggests that such events seem to require a familiar environment for them to develop, or that the real number of events is less than what has been declared. In all patients the recorded episodes arose from NREM sleep, as has been previously described in the literature [22].

Regarding pharmacologic treatments, there are case reports and small uncontrolled series which showed symptomatic benefits with the use of both topiramate and dopaminergic agonists [26–28]. In our series, 20 patients (58.8%) received topiramate in doses between 25 and 200 mg daily, showing satisfactory responses in most of them ($n = 17$; 85%) and an average follow-up time of 15.8 months. Of these patients, 30% had to withdraw their medication due to significant adverse effects, which is less than what has been reported in the literature. Winkelman [27] described 30 patients treated with topiramate, from which 68% were responsive for an average follow-up time of 11.6 months. The authors highlighted that 84% of their patients showed adverse effects and 28% lost more than 10% of their weight. Schenck and Mahowald [29] treated 17 patients, of which 12 were responsive and tolerated the medication well. Topiramate is an anticonvulsant agent that works through several mechanisms, including blocking sodium and calcium channel and inhibiting glutamate receptors (α-Amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid subtype) and carbonic anhydrase [30]; it also has GABAergic agonist properties. Some adverse effects are frequent, including dizziness, paresthesias, visual disturbances, kidney stones, excessive daytime sleepiness, and weight loss [30]. The adverse effects observed in the patients studied herein are common. The action mechanisms that would be useful for SRED patients are unknown, but they could be related to an anorexic or a sedative effect that helps patients sleep [24]. Other drugs used for SRED treatment include dopaminergic agents, such as levodopa, bromocriptine, and pramipexole [31–33]. Provini et al. [31] studied 11 patients through actigraphy measurement of sleep parameters and pramipexole (0.18–0.36 mg/day) for 2 weeks. The authors observed a decline in nighttime activity, but no changes in the number of awakenings or ingestion were observed. They concluded that the therapeutic effect would be seen in sleep quality improvements or motor activity reduction, more than having a direct effect on nighttime food ingestion. In our case series, only two patients used pramipexole, one of which responded well regarding both SRED and RLS, which the patient also had.

The SRED pathophysiology is unclear. It has been postulated as a dopaminergic dysfunction, based on the compulsive behavior that characterizes SRED episodes and on the response to dopamine treatment [34]. Another important feature of SRED is its frequent association with RLS, which suggests a common mechanism; usually RLS precedes SRED onset [31]. In addition, both RLS and SRED patients get symptomatic relief getting out of bed and food ingestion also can relieve RLS symptoms [31]. Tassinari et al. [35] have proposed that there is a release of some innate patterns of behavior in parasomnias controlled by central subcortical generators, which

would explain why some basic activities are seen during these such as walking or oroalimentary behaviors. In this regard, we can speculate that SRED could represent a release of alimentary behavior and that weight-centered anxiety frequently associated with this parasomnia could be a factor that contributes or enhances the SRED episodes.

Our series confirms previous studies which showed a frequent association between SRED and other sleep disorders, especially RLS, a condition which was found in 47% of our patients. In RLS there is a dysfunction of dopaminergic system and a suggestion of a possible functional impairment of mesolimbic structures [36], which could explain the compulsive nature of the behavior observed. Because the reward system is physiologically activated during sleep and sleep deprivation activates food intake behaviors, Perogamvros et al. [37] proposed that NREM sleep instability could be a contributing mechanism to the reward system dysfunction. It can be speculated that there is a release of the subcortical behavior control mechanisms in this scenario, as proposed by Tassinari et al. [35] for the parasomnias, particularly the oroalimentary parasomnia in this case. On this view, the integration of the 2 concepts (i.e., reward system dysfunction and release of innate behavior control mechanisms) could explain the link already known between SRED and RLS, but also with anxiety focused on weight.

5. Conclusion

Our series of 34 patients with SRED confirmed the clinical characteristics of the syndrome reported in the literature, including a predominance of women, a high association with sleep disorders and use of hypnotic drugs, and a satisfactory response to topiramate. It is worth noting the anxiety focused on weight as an additional element, which could give cues toward explaining the behavior focused on food ingestion of this parasomnia.

Conflict of interest

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: http://dx.doi.org/10.1016/j.sleep.2013.10.010.

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